ular (LV) remodelling after acute MI, but the mechanism of this improvement has never been assessed. We evaluated the relationship between ECFC levels and microvascular obstruction (MVO), and the impact of this relation on infarct size and LV remodelling at 6 months as assessed by magnetic resonance imaging (MRI).

Methods: 109 pts<75 years old, admitted with a first MI within 12 hours of onset of symptoms were enrolled. Peripheral blood samples were drawn to assess number of ECFC colonies (culture cells). Measurements of infarct size by MRI were performed at day 5 and 6 months.

Results: ECFC colonies were detected in 51/109 pts(47.2%) at admission ($ECFC^{pos}$ pts). At 5 days, MVO was more frequently observed (63% vs 33%; p=0.003) and of greater magnitude (7±6% vs 3±5%, p=0.0004) in $ECFC^{neg}$ patients versus $ECFC^{pos}$ pts respectively. At 6 months, there was a significantly greater reduction in infarct size in $ECFC^{pos}$ pts (–33.7±33.2% vs –15.1±24.6%, $ECFC^{pos}$ vs $ECFC^{neg}$ respectively; p=0.003). This reduction in infarct size was associated with a significant improvement in LV ejection fraction and a significant reduction in LV end diastolic and systolic volumes in $ECFC^{pos}$ pts. A significant positive correlation was observed among $ECFC^{pos}$ pts between MVO at day 5 and infarct size at 6 months (r^2=0.58, p<0.0001), while the number of ECFC colonies was significantly correlated with the relative change in infarct size at 6 months MRI (r^2=0.33, p<0.0001).

Conclusion: The presence of ECFC colonies is associated with a reduced degree of microvascular obstruction early after myocardial infarction, leading to reduced infarct size and positive LV remodelling at 6 months and can be considered as a marker of microvascular integrity in acute MI pts.

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Validation of assessment of circulate oxidative stress markers by the Free Oxygen Radicals Testing (FORT) assay among patients with an acute myocardial infarction.

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Background: Free oxygen radicals play an important role in the pathogenesis of many diseases including cardiovascular diseases, diabetes, cancer and aging. Several methods were developed for the direct or indirect measurement of oxygen free radical and its by-products. Using a new Free Oxygen Radicals Testing (FORT) the current study is designed first to validate the device and to investigate the potential relationships between the ROS and clinical or biological factors in human serum from a population of men with an acute myocardial infarction (AMI).

Methods: We first determined the effect of storage, variability and reproducibility of the FORT test in serum. Then we used the test in 66 patients from our bio bank of AMI patients.

Results: FORT values vary between 324 and 1198 FORT units, with a median value of 581 (494-754) FORT units. Among the risk factors, 17% of patients are diabetic, and 20% are obese. In univariate analysis, the FORT values seem to be influenced by age (r=0.161, p=0.195), presence of diabetes (p=0.102), a history of MI (p=0.181), LVEF <40% (p=0.005) and treatment with -blockers before admission (p=0.053), with ST-Elevation MI (p=0.058), levels of CRP (r=0.438, p<0.001), the rate of neutrophil (r=0.203, p=0.107) and peak CK (r=0.274, p=0.028). The analysis of multiple linear regression showed that CRP (p=0.023), LVEF <40% (p<0.001) and presence of diabetes (p=0.039) were independent predictors of serum FORT levels. This statistical model can explain 45% of the variance in the FORT levels.

Conclusions: The variability of the FORT on serum is minimal and thus reproducibility can be attained. FORT assay is stable when stored at 20 ˚C for a couple of months or at 4 ˚C for a few days. FORT correlation with CRP, LVEF and status of diabetes provides an interesting insight and a good link between oxidative stress and inflammation in patients with an AMI.

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The polymorphism Trp719Arg in the kinesin-like protein 6 is associated with the presence of late outgrowth endothelial progenitor cells in acute myocardial infarction

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Background: Much attention is focused on genetic polymorphisms associated with coronary artery disease. A candidate gene controversially associated with risk of coronary events is the kinesin-like protein 6 (KIF6), which is correlated with Trp719Arg polymorphism. In acute myocardial infarction (AMI), endothelial progenitor cells, particularly endothelial colony forming cells (ECFC) are mobilized from bone marrow and correlated with infarct size reduction. We investigated whether there was a relationship between presence of ECFC in AMI at admission and genetic status with regard to the KIF6 Trp719Arg polymorphism (rs20455).

Methods: Forty five patients aged <75 years old referred for a first STEMI or non STEMI AMI. Peripheral blood samples were drawn on admission. Isolated peripheral blood mononuclear cells were obtained by Hypaque-Ficoll density gradient centrifugation and cultured for 4 weeks. Cultured cells were phenotyped to assess the endothelial origin of ECFC. Genomic DNA was extracted in all patients and genotyping for allelic variations of KIF6 was performed

Results: Subjects were divided into two groups comparing the (Arg/Arg) homozygote variants with patients having a Trp allele. The genotype frequencies were 55%, 31% and 13% for Arg/Arg, Arg/Trp and Trp/Trp respectively. Between groups, higher levels of TnI, CK and CK-MB wer observed in the Arg/Arg group (respectively p=0.026, p=0.001 and p=0.031). ECFC were observed in 33% of patients with AMI. The percentage of patients without ECFC was significantly higher in the Arg/Arg group (p=0.033). No other significant differences were observed between groups.

Conclusion: In this report the Arg/Arg group showed a high number of ECFC-negative patients. A possible explanation might be the low mobilization of ECFC from bone marrow in this genotype since KIF6 is involved in cytokinesis. The altered amino acid Trp719Arg could decrease the ECFC released from the bone marrow in response to chemokines released at the onset of AMI.

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Renin-angiotensin-aldosterone system polymorphisms : a role or a hole in occurrence and long-term prognosis of acute myocardial infarction at Tunisian older people

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Background: Myocardial infarction (MI) is one of the major causes of death all over the world. Because MI frequently occurs suddenly without any preceding clinical symptoms, the prediction of MI is clinically of great importance. Angiotensin II is produced primarily by angiotensin I-converting enzyme (ACE) within atherosclerotic lesions and ACE level correlates with the severity of vessel wall damage. We analyzed the evolution with age of the frequencies of the I/D polymorphism of the ACE, A1166C of the angiotensin II AT1 receptor (AT1R), and M235T of the angiotensinogen (AGT) gene in a healthy population and he subsequent comparison to age- and sex-matched groups of MI patients.
Aim: To investigate the influence of increasing age on the incidence and remaining lifetime risk of myocardial infarction in a cohort of older men.

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